Estriol Assays to Assess Fetal Status

AMONG TESTS used to monitor fetal well-being in complicated pregnancies, estriol assays appear most useful. Estriol production rises a thousandfold from ovulation until term, and in late pregnancy depends overwhelmingly upon normal fetal as well as placental function. Hence, fetal distress will result in decreased estriol excretion even in conjunction with normal placental function. A normal pattern of 24-hour urinary estriols is regarded as a useful indicator of fetal well-being in pregnancies complicated by "postmaturity," intrauterine growth retardation, fetal death, hypertension and toxemia, and diabetes, if estriol is measured reliably, serially, and with appropriate frequency, particularly in diabetic women. Estriol excretion may be misleading in Rh-isoimmunization, and in anencephaly, as well as in cases with maternal corticosteroid and ampicillin therapy and impaired renal function. Falsely low estriol values may be due to mandelamine treatment, incomplete urine collection, and laboratory errors. Cumbersome and often inaccurate 24-hour urine collections may soon be obsolete when plasma estriol radioimmunoassays become generally available.

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Fibrin Split Products

COAGULATION SYSTEMS are activated *in vivo* in association with a variety of specific diseases. The presence in plasma of fibrin split products is one of the most sensitive indicators of an *in vivo* coagulative process (consumption coagulopathy). Fibrin split products are usually demonstrable in the disseminated forms of combined platelet and fibrinogen consumption characteristic of bacteremia, metastatic carcinoma and obstetric complications, as well as in more localized forms associated with venous thrombosis and tissue trauma. These forms of consumption coagulopathy appear relatively amenable to anticoagulation therapy with heparin. Fibrin split products are also usually demonstrable in consumption coagulopathy in which platelet consumption is dominant, as in arterial thrombosis, thrombotic thrombocytopenia purpura, hemolytic-uremic syndrome and vasculitic syndromes. These appear to be therapeutically more amenable to inhibitors of platelet function and the inflammatory reaction.

Fibrinogenolysis, the cleavage of fibrinogen—not fibrin—may occur secondary to consumption coagulopathy or may be independent. In this latter event, which appears to be associated with primary activation of the plasmin system, there is no platelet consumption, and fibrinogen split products are found in the plasma.

Hence with appropriate techniques that are now just emerging, it appears possible to detect *in vivo* coagulative processes as well as primary fibrinogenolytic processes. Diagnosis and management require rational integration of clinical and laboratory data.

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Carcinoembryonic Antigen (CEA) in Clinical Diagnosis

CARCINOEMBRYONIC ANTIGEN is a tumor-associated antigen that may prove useful in clinical diagnosis and management of malignant disease of the gastrointestinal tract and, to a lesser extent, the lung and breast. Low concentrations present in the plasma of normal persons are usually increased in patients with such diseases. The frequency of positivity and concentration correlates with the extent of tumor, being low in small nonmetastasized colonic carcinoma (40 percent positive in Dukes Stage A) and high in patients with metastatic tumor (90 percent in Dukes Stage D). Plasma CEA is elevated in more than half of patients with operable tumors (Dukes Stages A and B). The decline in plasma CEA following resection of carcinoma of the colon is of prognostic value. However, because of the complexity and differences between assays, results should be interpreted within the guidelines established by the laboratory.

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